RESEARCH PAPER:

Anti diabetic effects of *Ficus racemosa* on lipid profile in alloxan induced diabetic rats

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SUMMARY

The study was carried out to demonstrate anti diabetic effect of *Ficus racemosa* roots extract in alloxan induced diabetic rats with normal and control rats. The level of lipid (total cholesterol, triglyceardies, phospholipids and free fattyacids) significantly increased in diabetic rats as compared to control animals. The level of LDL and VLDL cholesterol significantly increased where as HDL- cholesterol level decreased in diabetic rats as compared to control animals. The results clearly indicate that aqueous and alcoholic extracts of *F. racemosa* roots at a dose of 400mg/kg/bw have shown anti hyperlipidimic in alloxan induced diabetic rats.

Key words :
Diabetes mellitus, *Ficus racemosa*, Cholestrol, Phospholipids

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Ficus racemosa is a medium tall tree with quite rich green foliage that provides good shade. It is popularly known as "Country fig" in English and "Atti" in Tamil. The leaves, bark and fruits of *F. racemosa* are employed in native medicine to treat several diseases (Joshi, 2000; Li *et al*., 2004). Experimental studies have demonstrated its anti-inflammatory, hepatoprotective and hypoglycemic effects (Mandal *et al*., 1999; Bhaskara Rao *et al*., 2002). However, there were no reports on antihyperlipidemic effect of *F. racemosa* root in alloxan-induced diabetic rats. In view of the above, it seems necessary to investigate the hypolipidemic activities of aqueous and ethanolic extract of *F. racemosa* root in alloxan-induced diabetic rats.

MATERIALS AND METHODS

Ficus racemosa roots were collected, cleaned, dried and powered. Both aqueous and alcoholic extracts were prepared. Diabetes mellitus was induced in wistar rats by single intraperitoneal injection of freshly prepared solution of alloxan monohydrate (150mg/kgbw) an in physiological saline after overnight fasting for 12 hrs (Gutteridge and Halliwell, 1990). A total of 35 numbers of rats were divided into 7 group and every group containing 1 animal. Group-1 animal served as control animal and did not receive any other treatment. Group-2 animals were provided single intraperitoneal injection of alloxan (150mg/kgwt) mono hydrate after over night fast 12 hrs. Group-3 and 4 animals received aqueous and alcoholic extracts of *F. racemosa* after the diabetic state was assessed. Group-5 animals received glibenclamide (600/mg/kgbw) for 45 days. Group-6 was provided oral administration of aqueous and alcoholic extracts of *F. racemosa* roots alone for 45 days. After the experimental period, all animals were sacrificed by cervical dislocation and biochemical studies were analyzed.

RESULTS AND DISCUSSION

The level of lipid (total cholesterol, triglyceardies, phospholipids and free fattyacids) were significantly increased in diabetic rats as compared to control animals. However, oral administration of aqueous and alcoholic extract of *F. racemosa* roots revert back the lipid profile values to near normal concentration in diabetic rats. No statistical significance was observed between control groups and rats treated with alcoholic and aqueous extracts of *F. racemosa* alone (Table 1).

The level of LDL and VLDL cholesterol significantly increased where as HDL-cholesterol level decreased in diabetic rats as compared to control animals. However, oral administration of aqueous and alcoholic extracts of *F. racemosa* roots revert back the lipoprotein values to near normal concentration in diabetic rats. No statistical significance was observed between control groups and rats

Table 1 : Lipid profile in plasma of control and experimental animals in each group

Groups	Cholesterol (mg/dl)	Triglycerides (mg/dl)	Phospholipids (mg/dl)	Free fatty acids(mg/dl)
1.control	82.8 ± 7.1	90.1 ± 5.9	84.7 ± 7.9	7.61 ± 5.3
2.Diabetic control	138.7 ± 11.5	166.9 ± 11.8	141.7 ± 12.8	12.54 ± 8.7
3.Diabetic + Aqueous *F. racemosa* roots	90.9 ± 10.4	97.8 ± 10.2	91.9 ± 5.7	8.22 ± 8.9
4. Diabetic + Alcoholic *F. racemosa* roots	92.7 ± 9.8	96.5 ± 6.9	90.2 ± 9.6	8.33 ± 6.2
5. Diabetic + glibenclamide	96.9 ± 7.6	100.3 ± 11.2	96.4 ± 11.2	8.71 ± 6.5
6. Control + Aqueous *F. racemosa* roots	84.2 ± 8.1	91.7 ± 10.3	86.2 ± 10.3	7.51 ± 4.8
7. control + Alcoholic *F. racemosa* roots	81.7 ± 8.3	92.1 ± 10.4	83.9 ± 10.4	7.74 ± 5.1

Values are expressed as mean ±SD (n=6); a – significantly different from control animals $^{a}p < 0.001$; b – significantly different from control animals $^{b}p < 0.01$; Non-significant

treated with alcoholic and aqueous extracts of *F. racemosa* alone (Table 2).

The level of lipids (cholestrol and phospholipids) significantly increased in diabetic rats as compared to control animals. However, oral administration of aqueous and alcoholic extracts of *Ficus racemosa* roots revert back the lipid levels in erythrocyte membrane to near normal concentrate in diabetic rats. No statistical significance was observed between control groups and rats treated with alcoholic and aqueous extracts of *Ficus racemosa* alone (Table 3).

The level of lipids (cholesterol, triglycerides, free fatty acids and phospholipids) significantly increased in diabetic rats as compared to control animals. Oral administration of aqueous and alcoholic extracts of *F. racemosa* roots revert back the lipid levels of lipids to near normal range in diabetic rats. No statistical significance was observed between control groups and rats treated with alcoholic and aqueous extracts of *F. racemosa* alone (Table 4).

The level of lipids (cholesterol, triglycerides, free fatty acids and phospholipids) significantly increased in diabetic rats as compared to control animals. Oral administration of aqueous and alcoholic extracts of *F. racemosa* roots revert back the lipid levels of lipids to near normal range in diabetic rats. No statistical significance was observed between control groups and rats treated with alcoholic and aqueous extracts of *Ficus racemosa* alone (Table 5).

As insulin has a profound role in the regulation of

Table 2 : Levels of lipoprotein in plasma of control and experimental animals in each group

Groups	HDL-C (mg/dl)	LDL-C (mg/dl)	VLDL-C (mg/dl)
1. Control	30.8 ± 2.7	42.7 ± 5.1	10.9 ± 1.1
2. Diabetic control	16.9 ± 2.1	120.8 ± 11.6	21.7 ± 2.3
3. Diabetic + Aqueous *F. racemosa* roots	26.7 ± 3.1	50.7 ± 4.8	12.8 ± 0.9
4. Diabetic + Alcoholic *F. racemosa* roots	27.2 ± 3.3	51.9 ± 5.3	13.1 ± 1.2
5. Diabetic + glibenclamide	24.5 ± 2.5	55.8 ± 6.1	14.2 ± 1.5
6. Control + Aqueous *F. racemosa* roots	30.1 ± 2.8	41.7 ± 4.8	10.4 ± 0.8
7. Control + Alcoholic *F. racemosa* roots	31.4 ± 2.2	40.9 ± 4.4	10.3 ± 0.6

Values are expressed as mean ±SD (n=6); a – significantly different from control animals $^{a}p < 0.001$; b – significantly different from control animals $^{b}p < 0.01$; NS – Non-significant

Table 3 : Lipid levels of erythrocyte membranes in control and experimental animals in each group

Groups	Cholesterol (µg/mg of protein)	Phospholipids (µg/mg of protein)
1. Control	138.6 ± 15.2	269.7 ± 18.9
2. Diabetic control	166.7 ± 18.9	261.6 ± 20.1
3. Diabetic + Aqueous *F. racemosa* roots	149.7 ± 16.7	265.8 ± 22.3
4. Diabetic + Alcoholic *F. racemosa* roots	147.8 ± 17.3	267.9 ± 19.7
5. Diabetic + glibenclamide	154.3 ± 14.2	262.6 ± 23.2
6. Control + Aqueous *F. racemosa* roots	140.2 ± 15.3	270.2 ± 18.6
7. Control + Alcoholic *F. racemosa* roots	136.9 ± 12.7	268.9 ± 19.9

Values are expressed as mean ±SD (n=6); a – significantly different from control animals $^{a}p < 0.001$; b – significantly different from control animals $^{b}p < 0.01$; Non-significant

Table 4 : Lipid profile in liver of control and experimental animals in each groups

Groups	Cholesterol (mg/100g tissue)	Triglycerides (mg/100g tissue)	Phospholipids (mg/100g tissue)	Free fatty acids (mg/100g tissue)
1. Control	300.6 ± 22.6	320.7 ± 26.9	1480.1 ± 130.9	590.8 ± 44.2
2. Diabetic control	451.9 ± 38.7	466.7 ± 38.7	2998.5 ± 270.1	887.6 ± 72.6
3. Diabetic + Aqueous *F.racemosa* roots	317.8 ± 24.9	330.8 ± 30.6	1494.3 ± 134.2	599.9 ± 60.1
4. Diabetic + Alcoholic *F.racemosa* roots	320.5 ± 25.8	329.6 ± 28.7	1488.7 ± 137.9	603.7 ± 54.9
5. Diabetic + glibenclamide	331.7 ± 28.7	339.7 ± 26.9	1510.2 ± 144.2	611.8 ± 57.2
6. Control + Aqueous *F.racemosa* roots	306.2 ± 24.2	316.8 ± 27.4	1476.1 ± 128.7	596.7 ± 45.4
7. Control + Alcoholic *F. racemosa* roots	304.4 ± 20.7	321.9 ± 25.2	1477.8 ± 126.3	592.3 ± 43.8

Values are expressed as mean ±SD (n=6); a – significantly different from control animals $^{a}p < 0.001$; b – significantly different from

Table 5 : Lipid profile in kidney of control and experimental animals in each group

Groups	Cholesterol (mg/100g tissue)	Triglycerides (mg/100g tissue)	Phospholipids (mg/100g tissue)	Free fatty acids (mg/100g tissue)
1. Control	415.8 ± 38.9	422.7 ± 40.2	1216.5 ± 115.3	349.6 ± 31.7
2. Diabetic control	667.6 ± 59.7	615.3 ± 66.9	3011.8 ± 290.8	554.8 ± 50.8
3. Diabetic + Aqueous *F. racemosa* roots	428.7 ± 36.2	438.7 ± 48.7	1232.6 ± 116.1	358.6 ± 29.6
4. Diabetic + Alcoholic *F. racemosa* roots	424.9 ± 35.9	434.6 ± 47.2	1228.7 ± 120.6	360.7 ± 31.4
5. Diabetic + glibenclamide	439.8 ± 40.1	443.7 ± 45.1	1240.8 ± 118.9	364.8 ± 35.2
6. Control + Aqueous *F. racemosa* roots	412.1 ± 34.8	418.6 ± 39.8	1211.9 ± 115.2	345.7 ± 31.9
7. Control + Alcoholic *F. racemosa* roots	416.7 ± 37.6	420.1 ± 38.6	1214.7 ± 110.9	350.1 ± 30.8

Values are expressed as mean ±SD (n=6); a – significantly different from control animals $^{a}p < 0.001$; b – significantly different from control animals $^{b}p < 0.01$; Non-significant

key enzymes involved in the lipid and lipoprotein metabolism, its deficiency causes major changes in the activity of these enzymes and thereby affecting overall lipid metabolism and lipid profile of various tissues (EL-Hazmi and Warsy, 1999). Insulin has also profound influence on the synthesis and expression of apolipoproteins in hepatic and extra hepatic tissues. Thus, the altered lipid and lipoprotein pattern observed in diabetic rats could be due to defect in insulin secretion and/or action (Okamoto, 1985; Sarti and Gallagher, 2006)

Hypercholesterolemia and hypertriglyceridemia have been reported to occur in alloxan-induced diabetic rats. Accumulation of cholesterol and phospholipids in liver due to elevated plasma free fatty acids has been reported in diabetic rats (Frayn, 2002; Mironava *et al*., 2000). The higher concentration of plasma total cholesterol observed in diabetic rats is probably due to mobilization of free fatty acids from the peripheral fat depots (Khan *et al*., 2003).

Alterations in the erythrocyte membranes lipid composition may be a reflection of alterations in the plasma lipid profile. HDL removes cholesterol from non-hepatic tissues to liver through the process known as reverse cholesterol transport. Several studies have documented reduction in plasma HDL cholesterol in diabetic rats and diabetic patients due to defect in reverse cholesterol transport (Das, 2003; Das and Baliarshinha, 1997). Our results support these observations.

Liver plays an important role in the catabolism and excretion of cholesterol. Profound increase in plasma and tissue lipids (cholesterol, phospholipids, triglycerides and free fatty acids) reported in diabetic animals. Triglycerides accumulation in the liver of diabetic rats is due to enhanced synthesis or decreased output from liver as VLDL or combination of both. The hypolipidemic effect of the *F. racemosa* roots extracts is due to inhibition of endogenous synthesis of lipids probably by potentiating the secretion of insulin (Krishnaswami, 1996).

In the present study was observed an increase in lipid parameters except HDL cholesterol in plasma, erythrocytes, liver and kidney of alloxan induced diabetic rats. It also indicates that the aqueous and alcoholic root extracts of *F. racemosa* have significant antihyperglycemic and antihyperlipidemic effects in alloxan induced diabetic rats. Further studies are warranted to isolate and characterize the bioactive antidiabetic principle from the roots of *F. racemosa*.

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